

L Number	Hits	Search Text	DB	Time stamp
1	78	cln3	USPAT; US-PPGPUB; DERWENT	2003/08/11 11:51
2	16	cln3 same (cancer\$6 or prolifer\$6 or overexpress\$6 or upregulat\$6)	USPAT; US-PPGPUB; DERWENT	2003/08/11 11:53
3	5679	boustany-\$.in. or guo-\$.in. or amalfitano-\$.in.	USPAT; US-PPGPUB; DERWENT	2003/08/11 11:54
4	364	(boustany-\$.in. or guo-\$.in. or amalfitano-\$.in.) and cancer	USPAT; US-PPGPUB; DERWENT	2003/08/11 11:54
5	3	((boustany-\$.in. or guo-\$.in. or amalfitano-\$.in.) and cancer) and cln3	USPAT; US-PPGPUB; DERWENT	2003/08/11 11:54

(FILE 'HOME' ENTERED AT 12:35:55 ON 11 AUG 2003)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:36:16 ON 11 AUG 2003

L1 1192 S CLN3
L2 118 S L1 AND OVEREXPRESS?
L3 17 S L2 AND (CANCER? OR PROLIFERATI?)
L4 7 DUP REM L3 (10 DUPLICATES REMOVED)
L5 205 S BOUSTANY R?/AU
E BOUSTANY R?/AU
L6 65 S E5-E6
L7 24 S L6 AND CLN3
L8 12 DUP REM L7 (12 DUPLICATES REMOVED)
E AMALIFTANO A?/AU

=>

L4 ANSWER 3 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:109700 BIOSIS
DOCUMENT NUMBER: PREV200100109700
TITLE: Blocking of **CLN3** expression by antisense
CLN3 adenovirus suppresses **cancer** growth
by modulating ceramide levels and delaying recovery from a
nocodazole induced G2M block.
AUTHOR(S): Rylova, S. (1); Jansen, P.; Amalfitano, A.; Pane, M.;
Boustany, R. M.
CORPORATE SOURCE: (1) Duke University Medical Center, Durham, NC USA
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No.
1-2, pp. Abstract No.-766.2. print.
Meeting Info.: 30th Annual Meeting of the Society of
Neuroscience New Orleans, LA, USA November 04-09, 2000
Society for Neuroscience
. ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Juvenile Batten disease is a neurodegenerative disease. Accelerated
apoptotic death of photoreceptors and neurons occurs due to defects in the
CLN3 gene. **CLN3** has antiapoptotic activity when
overexpressed in NT2 neuronal precursor cells. We have shown
overexpression of **CLN3** in a variety of **cancer**
cell lines and solid colon **cancer** tissue: **CLN3** is
overexpressed in glioblastoma (U-373G, T98g), neuroblastoma
(IMR-32, SK-N-MC), prostate (DU145, PC-3, LNCap), breast (BT-20, BT-549,
BT-474), colon (SW1116, SW480, HCT 116) and leukemia (HL-60) cell lines,
but not in malignant melanoma or pancreatic **cancer** lines. An
adenovirus bearing antisense **CLN3** (Ad-AsCLN3) was used to
transduce BT-20, SW1116, T98g **cancer** cell lines and resulted in
blocking of **CLN3** expression as seen by Western blot. Also
suppression of **cancer** cell growth was seen by 3H-Thymidine
incorporation and cell counting. Ceramide levels were increased 52% after
transduction of DU145 prostate **cancer** cells with 40 MOI of
Ad-AsCLN3 virus. Neuronal precursor NT2 stable cell lines both over and
underexpressing **CLN3** were synchronized by blocking them at the
G2/M phase of the cell cycle using Nocodazole. The cells
overexpressing **CLN3** rapidly exited G2/M and proceeded
through the cell cycle after removal of Nocodazole in comparison to NT2
cells underexpressing **CLN3** as seen by flow cytometry. Blocking
of **CLN3** expression using Ad-AsCLN3 suppresses growth of
cancer cells. This could be mediated by excess ceramide known to
inhibit cell growth and result in cell cycle arrest.

L4 ANSWER 1 OF 7 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002099667 MEDLINE
DOCUMENT NUMBER: 21818590 PubMed ID: 11830536
TITLE: The **CLN3** gene is a novel molecular target for
cancer drug discovery.
AUTHOR: Ryllova Svetlana N; Amalfitano Andrea; Persaud-Sawin
Dixie-Ann; Guo Wei-Xing; Chang Jerry; Jansen Paul J; Proia
Alan D; Boustany Rose-Mary
CORPORATE SOURCE: Department of Pediatrics, Duke University Medical Center,
Durham, North Carolina 27710, USA.
CONTRACT NUMBER: R01 DK 52925 (NIDDK)
R02 NS 30170 (NINDS)
SOURCE: CANCER RESEARCH, (2002 Feb 1) 62 (3) 801-8.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020207
Last Updated on STN: 20020307
Entered Medline: 20020305

AB Juvenile Batten disease is a neurodegenerative disease caused by
accelerated apoptotic death of photoreceptors and neurons attributable to
defects in the **CLN3** gene. **CLN3** is antiapoptotic when
overexpressed in NT2 neuronal precursor cells. **CLN3**
negatively modulates endogenous ceramide levels in NT2 cells and acts
upstream of ceramide generation. Because defects in regulation of
apoptosis are involved in the development of **cancer**, we
evaluated the expression of **CLN3** on both mRNA and protein levels
in a variety of **cancer** cell lines and solid colon **cancer**
tissue. We also observed the effect of the blocking of **CLN3**
protein expression on **cancer** cell growth, survival, ceramide
production, and apoptosis by using an adenovirus-bearing antisense
CLN3 construct. We show that **CLN3** mRNA and protein are
overexpressed in glioblastoma (U-373G and T98g), neuroblastoma
(IMR-32 and SK-N-MC), prostate (Du145, PC-3, and LNCaP), ovarian (SK-OV-3,
SW626, and PA-1), breast (BT-20, BT-549, and BT-474), and colon (SW1116,
SW480, and HCT 116) **cancer** cell lines but not in pancreatic
(CAPAN and As-PC-1) or lung (A-549 and NCI-H520) **cancer** cell
lines. **CLN3** is also up-regulated in mouse melanoma and breast
carcinoma **cancer** cell lines. We found **CLN3** expression
is 22-330% higher than in corresponding normal colon control tissue in 8
of 10 solid colon tumors. An adenovirus-expressing antisense **CLN3**
(Ad-AS-**CLN3**) blocks **CLN3** protein expression in DU-145,
BT-20, SW1116, and T98g **cancer** cell lines as seen by Western
blot. Blocking of **CLN3** expression using Ad-AS-**CLN3**
inhibits growth and viability of **cancer** cells. It also causes
elevation in endogenous ceramide production through de novo ceramide
synthesis and results in increased apoptosis as shown by propidium iodide
and JC-1 staining. This suggests that Ad-AS-**CLN3** may be an
option for therapy in some **cancers**. More importantly these
results suggest that **CLN3** is a novel molecular target for
cancer drug discovery.

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RESULT:

1 GeneCard matches your *precise* query for "cln3";

It is represented by a minicard.

Click "Display" on the left to get the full GeneCard.

|||Description=ceroid lipofuscinosis, neuronal 3, juvenile (Batten, Spielmeyer-Vogt disease)
|||Similarity=80.54|LocusLink=12752|Unigene=232156|GenBank=NM_009907.1|||Organism=XI|Symbol:
|||Description=ESTs, Weakly similar to A57219 Batten disease-related protein **CLN3** - human [H.sapien]
|||Similarity=75.65|LocusLink=|Unigene=84969|GenBank=BJ043920.1|||

[This Search Engine uses glimpse and Excite technology]

Developed at the Crown Human Genome Center & Bioinformatics Unit, at the Weizmann Institute of Science

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National Library of Medicine - Medical Subject Headings

2003 MeSH

MeSH Supplementary Concept Data[Return to Entry Page](#)

Name of Substance	Batten disease protein CLN3
Record Type	C
Registry Number	0
Entry Term	CLN3 gene product, human
Entry Term	CLN3 protein, human
Entry Term	ceroid lipofuscinosis, neuronal 3 protein
Entry Term	CLN3 protein, Batten disease
Entry Term	battenin
Heading Mapped to	*Proteins
Indexing Information	Neuronal Ceroid-Lipofuscinosis
Source	Genomics 1997 Mar 1;40(2):346-50
Frequency	51
Note	base sequence in first source; GenBank U32680; mouse homolog = CLN3 PROTEIN, MOUSE; don't confuse with CLN3 PROTEIN, YEAST
Date of Entry	19970421
Revision Date	20030228
Unique ID	C105199

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